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LONGITUDINAL ASSESSMENT OF DISEASE SITES BY ATTACHMENT LEVEL CHANGES AND BONE DENSITY LOSS BY DIGITAL IMAGE ANALYSIS

A THESIS

Presented to the Faculty of

The University of Texas Graduate School of Biomedical Sciences

at San Antonio

in Partial Fulfillment of the Requirements for the Degree of

MASTER OF SCIENCE

By

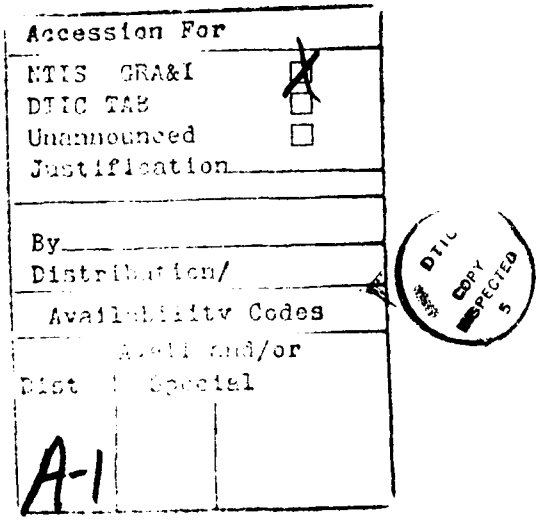
David E. Deas D.M.D

San Antonio, Texas

April 1989

DEDICATION

I dedicate this thesis to my family for all of the encouragement and support they've given me over the years. This includes my parents, Frank and Audrey Deas, who made the education of their children a top priority, and my children, Steven, Andrew, and Laura, who have loved me in spite of my unavailability during this residency. Above all, I thank my wife Susan for her tremendous patience and understanding during the course of this project. Her love and friendship have made these three years not only bearable but fun, and as long as she is by my side I look forward to whatever the future may bring.



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LONGITUDINAL ASSESSMENT OF DISEASE SITES BY ATTACHMENT LEVEL CHANGES AND BONE DENSITY LOSS BY DIGITAL IMAGE ANALYSIS

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There are currently no definitive methods for clinically assessing initiation or progression of periodontitis. Repeated attachment level measurements have been used by several investigators, with a change of $\geq$ 2mm being indicative of 'true' progression. Other recent studies have established digital subtraction radiography as a sensitive method of

detecting even slight density change within alveolar bone. This study attempted to evaluate such changes in radiographic density as a potential indicator of disease progression. Twenty-one patients with a history of periodontitis were monitored using both clinical and radiographic means. Of these patients, 10 were untreated, 3 had been treated and received active maintenance care during the monitoring period, and 8 were treated but received no maintenance care. Measurement intervals were at baseline, 3, 6, and 9 months.

Clinical parameters included duplicate measurements of probing depth and probing attachment level recorded to the nearest millimeter at interproximal sites adjacent to all teeth. For each arch, a pressure molded vacuform stent provided a reproducible reference for probing. Measurements were made using standardized 20mm probes with a tip diameter of 0.4mm. All probing measurements were made by a single examiner at approximately 25 grams of force. For statistical analysis, the mean value of duplicate attachment level measurements was considered the definitive measurement.

Radiographic data was compiled from standardized D speed vertical bite wing radiographs made of posterior sextants at each time interval. Film position was assured by film holders modified with vinyl polysiloxane occlusal registration. Head position was maintained by a cephalostat. Exposure settings were 80 KVP, 100 mA, 1/2 sec. Radiographs were analyzed by computer assisted densitometric image analysis (CADIA), which monitored density change within pre-determined areas of interdental bone. Density change was recorded in CADIA units, which were derived from both the area and magnitude of density change. These values were recorded as density loss (degeneration), density gain (regeneration), and overall net change.

Results indicate that the majority of probing sites exhibited no

attachment level change during the nine month period; however the percentage of sites with attachment loss increased with time. At nine months, the untreated and treated/no maintenance groups exhibited similar amounts of attachment loss, while treated/maintenance subjects exhibited proportionately less. For untreated and treated/no maintenance groups, a higher percentage of attachment loss was noted at sites initially deeper than 3mm; attachment loss was more frequently seen in the treated/maintenance group at sites initially 3 mm or less.

Due to the two dimensional nature of radiographs, one area of alveolar bone available for density analysis represented up to four probing sites. Density analysis was therefore calculated in terms of radiographic 'complexes' of multiple probing sites. When density change at complexes with > 2mm of attachment loss was compared to complexes without significant attachment loss, both groups were found to experience a net degeneration. Mean degeneration at attachment loss sites was significantly greater at 9 months, but not at 3 or 6 months. This suggests that even though density and attachment loss may occur simultaneously, this study was unable to demonstrate that density loss could predict future attachment loss. Also, density loss tended to increase as more sites within each complex experienced attachment loss.

It was concluded that a significant correlation existed between mean density and attachment level changes during the same time interval; however, there were wide variations at individual sites. Also, as evaluated in this study, there appeared to be little value of monitoring density change to predict future episodes of attachment loss. Studies such as this one are hampered by comparison to the inadequate 'gold standard' of probing attachment level change.

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I. Introduction and Literature Review

A. Progression of Periodontal Disease

The literature is replete with studies describing the progression of periodontal disease. It was long accepted that if untreated, diseased sites would undergo a progressive rate of breakdown that resulted in a steady loss of attachment. Information compiled by various epidemiological surveys seemed to suggest that periodontal disease is a chronic progressive disorder that worsens with age (Marshall-Day et al 1955, Bossert and Marks 1956, Johnson et al 1965, Kelley and Harvey 1977). A longitudinal study by Loe et al (1978) investigated the rate of progression of periodontal disease in 2 specific population groups over a period of 8 years. Their initial conclusion supported the concept that the destruction of periodontal tissues progresses steadily over time.

Observations of individual cases, however, often failed to confirm this theory. Patients with untreated periodontitis were found to vary widely with regard to rate of progression. Moskow (1978) described a woman with advanced periodontitis that did not progress over a 10 year period. A longitudinal study of untreated patients (Becker et al 1979) reported that disease progression varied widely in the same mouth. A more recent longitudinal study of untreated (but frequently monitored) patients reported that sites which developed disease over one period of time did not necessarily deteriorate further (Lindhe et al 1983). Haffajee et al (1983) reported a study of 22 patients with untreated periodontal disease. They examined

3,414 tooth surfaces for one year, and determined that only 3% of those sites experienced significant attachment loss. Finally, the latest report of the longitudinal survey by Loe et al (1986) identified three subpopulations of patients with periodontal disease: 1) patients with rapid progression - 8%, 2) patients with moderate progression - 81%, and 3) patients with no progression - 11%. Also, it was noted that within these groups, disease progression at different sites varied widely.

Current theories of disease progression reflect the inconsistency of activity found by these examiners. Socransky et al (1984) proposed that disease progression occurs in recurrent, acute episodes. In addition, disease activity at certain sites can progress faster or slower than can be predicted from estimates of prior disease activity. To account for these observations, the authors described disease progression as 'bursts' of activity occurring for short periods of time in individual sites. Some sites appear to undergo a brief active burst of destructive disease followed by a period of remission. Other sites may undergo continual disease activity resulting in extensive attachment loss over a short period of time. Still other sites may remain free of disease throughout the patient's life.

B. Methods to Determine Disease Activity

With such a model of periodontal disease, it is obviously necessary to have an accurate method to determine active sites. The clinical parameters of bleeding on probing, pocket depth, redness, increased crevicular fluid volume, plaque accumulation and suppuration have all been used to determine

disease activity, but recent literature has raised questions about their effectiveness. It appears that while clinical parameters may be useful to describe certain characteristics and classify patients into groups, they are not useful in identifying microbiological or immunological differences, nor can they identify areas at risk for future disease (Kornman 1987). The limitations of clinical parameters was probably best demonstrated by Haffajee et al (1983). They performed periodic measurements using these parameters as predictors of future attachment loss. They concluded that none of these indicators, used either alone or in combination, was an accurate predictor of future disease activity.

1. Probing Attachment Level Measurements. Currently, disease activity is usually identified by comparing clinical attachment level measurements over various time periods. Although probing measurements have for years been used to monitor periodontal disease, probing has been associated with large amounts of variability. Listgarten (1980) reviewed some of the problems associated with probing measurements. The probe tip may not reach the base of the sulcus in a healthy site, and may penetrate the base of the sulcus in an inflamed site. The angulation of the probe, shape of the tooth, and presence of subgingival calculus can all affect probing measurements. Other investigators have reported non-reproducibility of measurements based on probing force (Abbas et al 1982, Kalkwarf 1986, Van der Velden 1980) and location of sites within the arch (Watts 1987, Freed et al 1983).

Due to changes in gingival tone and contour, the position of the gingival margin may change significantly depending on the state of

inflammation. This potential difference in gingival height from one time period to the next adds still another potential source of probing error. To avoid this problem, the practice of recording probing measurements from a fixed reference point has been adopted for use in clinical studies (Ramfjord 1959). The cemento-enamel junction is commonly used for this purpose (Goodson 1982), but in cases of abrasion, cervical restoration, or short clinical crowns the CEJ may be hard to identify.

One commonly used alternative is the onlay stent, which provides a well defined supragingival reference point from which probing measurements can more easily be made. Many variations of probing stents have been described in the literature. Hellden et al (1979) used vacuum adapted stents to monitor attachment levels in a group of patients treated with varying combinations of scaling and antibiotic therapy. A similar technique was used by Watts (1987) in a study of constant force probing. Badersten et al (1981) used metal onlays to study attachment level changes following non-surgical therapy. Isador and Karring (1984) used flexible silicone stents to examine the reproducibility of pocket depth and transgingival probing measurements. Finally, Clark et al (1987) compared the reproducibility of duplicate attachment level measurements made using both the CEJ and Duralay acrylic stents as fixed reference points. Higher correlation coefficients were found for both inter and intra-examiner reliability when stents were used.

2. Statistical significance of attachment level measurements. To account for the potential errors inherent in the probing technique, various groups have established thresholds for 'significant' attachment level change.

Investigators from Forsythe (Haffajee 1983-b), for example, used the statistical variability of repeated probing measurements to suggest that the loss of attachment identifying progressing disease is greater than or equal to 2 mm. This threshold value has subsequently been used in any number of studies monitoring attachment level measurements, such as those by Lindhe (1983), Lindhe and Nyman (1984), and Lang (1986). This approach greatly limits the chances of reporting disease activity where it does not exist (false positives), but also very likely fails to identify disease progression that occurs within the 2 mm range of variability (false negatives). Even though the statistical interpretation of probing attachment level measurements has been criticized in recent years (Imrey 1986), at this time no one has established a more reliable technique. For this reason, in spite of its shortcomings, the longitudinal assessment of attachment level measurements remains the 'gold standard' for the progression of periodontal disease.

C. Radiographic Assessments of Disease Progression.

1. Conventional Radiographic Technique. Since changes in the alveolar crestal bone most certainly occur in periodontitis, the analysis of sequential radiographs should also offer a means to monitor disease progression. One problem with this approach is that the diagnostic sensitivity of radiographic parameters to assess disease is rather uncertain. Using conventional radiographic techniques, Hollender et al (1966) were unable to find evidence of osseous changes in a group of children with severe gingivitis. Similarly, neither Ramadan and Mitchell (1966), nor Ainamo and

Tammisalo (1973) were able to demonstrate early crestal bone loss in radiographs. Studies such as these suggest that standard radiography is of limited value in assessing the initial destructive changes in the periodontium.

Other authors have investigated the diagnostic sensitivity of radiographs by determining the size, shape and position of bony lesions that can be visualized in radiographs. Several studies using created defects in dried skulls have reported that interproximal lesions were not visible as long as the cortical plates remained intact (Bender and Seltzer 1961a,b, Goldman 1957, Ramadan and Mitchell 1962). Another report stated that experimentally induced septal defects were noticed radiographically only if they had a minimal depth of 3 mm (Pauls and Trott 1966). Using more standardized techniques of film positioning, exposure, and processing, Shoha et al (1974) were able to observe septal defects in premolar regions without involvement of the cortical plates. It is possible that the thinner cortical bone in this region allowed more film contrast and better visualization of defects.

Another difficulty in using radiographs to monitor disease progression is the poor correlation between clinical and radiographic parameters. Greenstein et al (1981) reported no association between clinical findings and the radiographic appearance of the lamina dura. Ainamo and Tammisalo (1973), in a study of young adult males, were unable to relate the gingival index (GI) to differences in the width of the periodontal space, continuity of crestal bone, or patterns of trabeculation. Finally, Mann et al (1985),

using the same radiographic criteria, were unable to correlate findings from bite-wing radiographs to the clinical parameter of attachment loss. Each of the previous studies suggested that conventional dental radiographic technique does not offer the diagnostic ability to monitor disease progression over short periods of time.

2. Imaging Techniques in Dental Radiography. A possible explanation for the poor performance of radiographs as indicators of disease progression is that the human eye is rather insensitive to small radiographic changes. If this is indeed true, it is also likely that the radiographic image may contain useful information that the investigator simply can not see. In recent years, new imaging methods have become available that can reveal early density changes in the hard tissues of the oral cavity. The sensitivity of these new methods has encouraged researchers to continue to investigate the relationship of the radiographic appearance of alveolar bone to the activity of periodontal disease.

The first imaging technique used for this purpose was subtraction radiography, which was initially used in medicine to study the image of blood vessels in arteriography. The subtraction technique requires a pair of sequential standardized radiographs. From the baseline film, a 'mask' is produced on a film duplicator. The subsequent radiograph is placed on top of the 'mask', and a subtracted image is obtained on the film duplicator. The mask serves to filter all of the similar features of the 2 films; therefore the subtracted image consists of only those small portions of the radiograph where there is actual change. This technique was utilized by Lurie et al

(1983) to demonstrate early loss of alveolar bone in ligature induced periodontitis in a monkey model. Subtraction radiography has also been used in conjunction with television equipment that produces a visual subtraction readout (Klein 1967). This technique was used to investigate the early formation of periapical lesions prior to diagnosis by clinical methods (Kassle and Klein 1976).

The value of subtracted images to a definitive radiographic diagnosis was reported by Grondahl et al (1987). They found that both inter and intra-examiner agreement rates were significantly better when subtracted images were evaluated rather than the individual films. The same group later reported that subtracted images could identify created defects in dried skulls with an accuracy unmatched by conventional radiographs until the defects were three times deeper (Grondahl et al 1988). Both of these studies suggest that subtraction radiography makes the observer more sensitive to slight changes at the alveolar crest.

Conversion of dental radiographs into a digital format was first described by Ando (1969). This process consisted of scanning the radiograph at 5400 - 5600 picture points with a microphotometer, then assigning the picture points a grey level value from 0 to 255. This data was processed mathematically, and relative changes in the picture points were used to monitor density changes in a treated periodontal case. This technique was further improved by the introduction of video cameras for viewing radiographs and histological sections (reviewed in Bragger 1988a).

The combination of the video and computer technology required for digital imaging and subtraction radiography in medical research led to the development of a digital subtraction technique for dental radiography. The initial studies using this method sought to identify qualitative differences in radiographic images that could be visually identified in a manner similar to conventional subtraction radiography. Groendahl et al (1983) used a video based image processor to measure the light intensity transmitted at each picture point (pixel) in a series of standardized dental radiographs. The transmitted light was converted to gray level values ranging from 0 to 63; the digitized image was then stored in a computer and displayed on a TV monitor as a positive image. The subsequent experimental radiograph was displayed as a negative image, and was aligned to the structures present in the baseline film. Differences in density between the 2 films were identified as lighter (gain in density) or darker (loss of density) areas against a gray background.

Digital subtraction radiography has also been used in an attempt to quantitate the volume of osseous lesions present in dental radiographs. Ruttiman (1985, 1986) produced cylindrical lesions in dried skulls. A continuous bone wedge with known dimensions was superimposed on the baseline radiograph, so that a linear gray scale wedge appeared on the subtracted image. The outline of the lesion was traced to quantify the length and width, while the depth of the lesion was calculated from the superimposed gray level scale. Comparison of the actual volume with the calculated volume gave agreement within 10%.

Two laboratory comparisons of digital subtraction radiography and 125^{I} absorptiometry found that both methods have similar sensitivities for detection of osseous defects in dried skulls (Ortman et al 1985, Grondahl et al 1988). A similar comparison was reported by Hausmann et al (1981), using the 'Magiscan' system to digitize radiographic images in a 64 step grey level scale. The authors reported a correlation coefficient of 0.96 between the 2 methods, indicating that the digital subtraction technique was a sensitive indicator of bone density change. A clinical evaluation of this same system (Hausman et al 1985) suggested that density change as determined by digital subtraction radiography is a more sensitive method to monitor crestal bone than are changes in crestal bone height.

A further refinement of this computer based video technology is the computer-assisted densitometric image analysis (CADIA) method, which was developed to quantitatively assess alveolar bone density changes. The CADIA system was tested in several studies by Bragger et al (1988b,c). Standardized serial vertical bite wing radiographs were taken using film holders with a built-in step wedge. The radiographic images were processed according to a range of grey levels with numerical values from 0 to 255 and stored in a main frame computer. An algorithm was developed to correct for slight density changes due to exposure and developing differences between baseline and subsequent experimental films. Windows of interest within the alveolar bone were outlined with a cursor on the baseline image; the densitometric change in these areas on experimental films was quantitatively recorded for a printed report. The units for reporting density change, called CADIA units, were derived by multiplying the area affected by density

change by the magnitude of density change. An in vitro analysis found a highly significant correlation in decalcified bone specimens between CADIA assessed density change and actual calcium loss as determined by atomic absorption spectroscopy (Bragger 1988b).

A clinical trial of the CADIA system was used to assess density changes at bone sites exposed to surgical procedures. The CADIA system was able to identify surgically induced bone loss with a sensitivity of 82%, a specificity of 88%, and a diagnostic accuracy of 87%. This was far more accurate than a clinical examination of radiographs by a group of experienced periodontists (Bragger et al 1988b). In a later report, this group used CADIA analysis to monitor the post surgical remodelling of alveolar bone at these same sites (Bragger 1988c).

3. Image Analysis to Determine Disease Progression. The documented sensitivity of this new radiographic technology offers additional opportunities to investigate changes in alveolar bone with the progression of periodontitis. The first published report of a study using these techniques to monitor disease progression was published by Hausmann et al (1986). Utilizing digital subtraction radiography, the authors monitored a group of 15 patients with untreated periodontitis for a period of 6 months. They found that 9% of observed areas of crestal bone exhibited bone loss, while 4% of the observed sites exhibited bone gain. Nine of the 15 subjects showed evidence of bone loss, 2 showed both gain and loss, and 1 showed gain only. The clinical parameters of pocket depth, gingival index, and plaque index were of little value in predicting the bone changes seen by subtraction

radiography. The authors concluded that the observed pattern of crestal bone changes was consistent with the episodic model of disease progression. This pattern of bone change was also seen in a study utilizing 125^{I} absorptiometry to monitor patients with untreated disease (Hausmann and McHenry 1982). A more recently reported animal study used digital image analysis to investigate bone changes in experimental gingivitis (Ericsson et al 1988). After 3 weeks of plaque accumulation, the authors found no evidence of bone density changes at sites with histologically documented gingivitis.

D. Statement of Problem.

The purpose of the present investigation is to examine evidence of disease progression in periodontitis patients as determined by CADIA analysis of alveolar bone and by attachment level change. In this manner we hope to evaluate quantitative changes in radiographic density as a more sensitive indicator of disease progression. It is possible that the incidence of active sites may be higher than observed with the parameter of attachment level change. Also, by longitudinally evaluating these subjects, we should be able to examine the relationship between bone loss and attachment level change. The current theory regarding this relationship was proposed by Goodson (1984) in an investigation of changes in bone height in untreated patients. He determined that attachment loss is often followed by a period of remission in which the attachment is reformed at a more apical level. Concurrently, radiographic evidence of bone loss is seen in several months as the alveolar bone remodels to this more apical position. Using the more sensitive criteria of density change rather than change in bone height, it is

possible that attachment loss is much more closely associated with changes at the alveolar crest. Finally, we hope to investigate whether disease activity can be radiographically identified prior to the clinical observation of attachment loss. If so, it may be possible to better determine the specific treatment needs of the patient.

II. Materials and Methods

A. Selection of Subjects

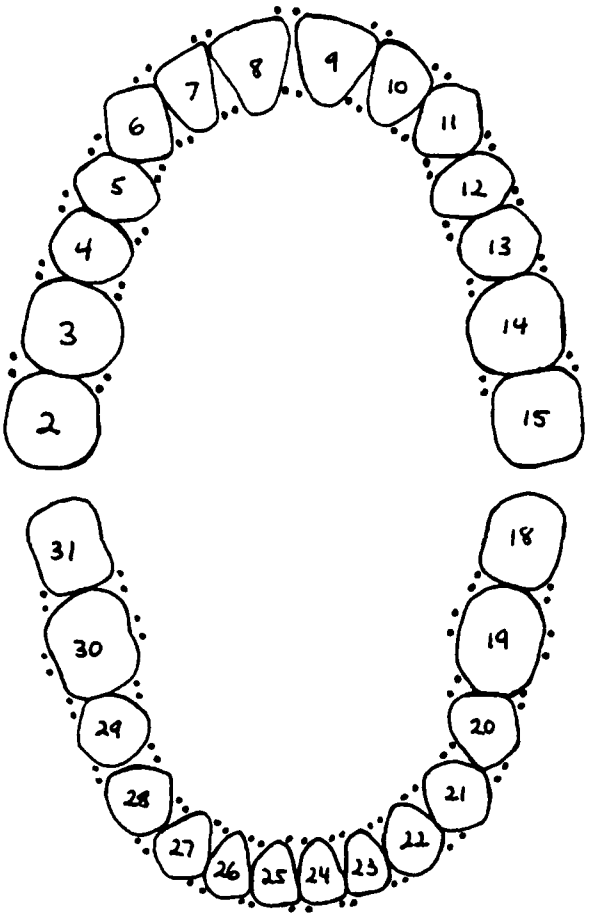
Twenty - five subjects with a history of periodontitis participated in this study conducted at the University of Texas Health Science Center at San Antonio (UTHSCSA). Approximately 18 of these subjects were initially contacted based on their interest in a previous study. The remaining 7 were identified from a list of patients who had been previously treated in the post-graduate periodontics clinic. All subjects were selected on the basis of a screening appointment, which consisted of a health history review and an oral examination for the presence of significant probing depths and attachment loss. Pregnant women and those patients with uncontrolled systemic disease were not considered. Individuals found to be acceptable for study purposes were invited to participate in a study investigating the progression of 'gum disease' using both 'gum measurements' and X-Rays. A consent form approved by the UTHSCSA Institutional Review Board was reviewed and signed by each subject. From these 25 subjects, clinical and radiographic data was recorded at baseline, 3, 6, and 9 months.

B. Clinical Data.

The clinical data consisted of duplicate probing attachment level (PAL) measurements recorded at interproximal sites adjacent to each tooth. Four additional sites were probed adjacent to mandibular first molars in order to examine evidence of attachment loss in furcations (See Figure 1). In

Figure 1.

Probing sites. Interproximal probing measurements were made on both facial and lingual surfaces of all teeth except third molars. Distal surfaces of second molars were not reliably present in radiographs and therefore not included. Four additional sites were measured on the buccal and lingual surfaces of mandibular first molars in order to examine evidence of attachment loss in furcations.



PROBING SITES

addition, the presence or absence of bleeding on probing was noted at each site. All probing measurements were taken by a single examiner at approximately 25 grams of probing force. The probe used in this study was the 'Loma Linda 20' (Hu Friedy) with a tip diameter of 0.4 mm. Probing force was calibrated at monthly intervals against a metric scale. A reference for probing measurements was provided by maxillary and mandibular occlusal stents fabricated in the following manner.

Following the screening examination, alginate impressions were taken of each subject to provide maxillary and mandibular diagnostic casts. Clear plastic stents were formed on these casts using a pressure molded vacuform matrix technique (Biostar). The vacuform stents were trimmed at a level several millimeters above the gingival margin, and the apical border was traced with a permanent marker to increase visibility. These stents were used to obtain reproducible attachment level measurements in a manner similar to the method described by Isador et al (1984). At each site, pocket depth to the nearest millimeter was recorded first, then the distance from the gingival margin to the stent. These values were added to give the value of probing attachment level (PAL). At each three month interval, probing measurements were first recorded at each site. The patient was then taken to a separate clinic for radiographs prior to the second set of probing measurements. This made the time interval between measurements about 40 minutes. For purposes of statistical analysis, the mean value of the duplicate attachment level measurements was used as the definitive value. Probing attachment level change was examined at threshold values of >1mm and > 2 mm at each three month period.

C. Radiographic Data.

The radiographic data was compiled from D speed vertical bite wing radiographs taken of all posterior sextants. The radiographs were standardized using film holders to maintain film position and a cephalostat to maintain head position (Jeffcoat et al 1986). A single X-Ray machine (Gerdex- General Electric) was used to expose all radiographs; exposure settings of 80 KVP, 100mA, 1/2 second were determined from preliminary investigation to give the best film density for computer image analysis. The X- Ray film was from a single lot of Kodak film provided by the manufacturer. Each film was processed in a manual developing system which was monitored daily for temperature and chemical solution activity.

Vertical bite wing film holders (Rinn Co.) were customized by adding an occlusal registration of polyvinylsiloxane impression putty (Exaflex). Depending on the arch size and number of teeth, each subject had from 2 - 5 film holders customized in this manner to obtain standardized radiographs of all posterior teeth. The angulation of the patient's head within the cephalostat to make each film perpendicular to the X-Ray beam was recorded for each film position. The customized film holders were stored with diagnostic casts and probing stents in sealed plastic containers at room temperature.

Each radiograph was analyzed using Computer Assisted Densitometric Image Analysis (CADIA). This was accomplished by transferring the film onto a

TV-monitor (Grinnel Systems) by means of a film camera (Eyecom). The radiographic image was digitized according to a non-parametric grey level standardization program (Ruttiman et al 1986) and stored in a mainframe computer (Digital Systems) according to a grid of 512 x 480 picture points or pixels. In this manner, the radiographic image was stored for mathematical computation as well as recalled to allow viewing on the television monitor. For each baseline image, the edges of teeth and metallic restorations were outlined automatically and stored for the purpose of orienting subsequent experimental films.

Specific windows of interest within baseline radiographs were established using a cursor to outline the desired areas. For this study, windows of interest included the alveolar crest in interproximal regions, the furcation areas of mandibular first molars, and at least one area of dentin to provide a negative control. In addition, 2 distinct points on each radiograph (usually interproximal contacts or metal margins) were located with the cursor; the distance between these 2 points was recorded in millimeters and used to determine the area of subsequent density change.

Experimental films taken at 3, 6, and 9 months were compared to each baseline image. Edges established for the baseline film were reproduced on the monitor, allowing the experimental films to be properly aligned. In the windows of interest, the average grey levels of all 2 x 2 pixel areas in the baseline and experimental images were subtracted. In order to exclude background noise that could produce false positive readings of density change, only pixel areas which changed more than 7 grey levels were recorded.

Grey level differences representing density change were calculated and recorded for a printed report. For each window, numerical values for increased density, loss of density, and the overall net change were recorded. These values were given in CADIA units, which were derived by multiplying the area of density change by the mean difference in the grey level averages described above. For purposes of description, density loss is referred to as degeneration, while increased density is referred to as regeneration.

The clinical and radiographic data was combined to provide information relative to the following topics: 1) Probing attachment level change, 2) radiographic density change, and 3) correlation between attachment level and density change.

III. Results

A. Treatment Groups

Of the original 25 subjects, only 21 participated throughout the length of the study period. Even though each of these 21 subjects had a documented history of pocket formation and attachment loss, three distinct groups were identified based on previous exposure to periodontal therapy. Ten subjects had never before been treated for periodontitis and were classified as untreated. Of the remaining eleven, all but two had been surgically treated at times ranging from two to seven years prior to the study. Three of these subjects received recall maintenance care during the study period and therefore comprised a treated with maintenance group, while the other eight either received no maintenance care or were classified as refractory to periodontal therapy. These eight subjects comprised a treated with no maintenance group. From these 21 subjects we were able to observe 2,094 sites over the 9 month period. Initial probing depths from these sites indicated that the untreated subjects had a significantly higher percentage of deeper sites than the other 2 groups (Table 1).

B. Probing Attachment Level Data.

The first aspect of the probing attachment level data concerned the reproducibility of attachment level measurements. Based on 8,076 duplicate measurements recorded from 2,094 sites, reproducibility $\pm$ 1 mm was 95.8%, while reproducibility $\pm$ 2 mm was 99.6%. This is within the range of other studies using stents to determine attachment level measurements, such as the

Table 1: Pocket Depths at Baseline. The percentage of sites with initial pocket depths ≤ 3 mm, 3.5 mm - 6.0 mm, and > 6mm are listed. Note that Untreated subjects had a proportionately higher number of deeper sites.

GROUP	≤ 3 mm	3.5 - 6.0 mm	> 6mm
UNTREATED	64.4%	31.3%	4.3%
TREATED/ NO MAINT	88.2%	11.8%	0.0%
TREATED/ MAINT	88.7%	11.3%	0.0%

one published by Isador and Karring (1984).

If data from all patients is examined collectively, it is evident that the vast majority of sites did not exhibit a significant attachment level change at any of the three measurement intervals. If changes >1 mm are examined (Table 2), 13.5% of all sites exhibited attachment loss at nine months compared to 1.4% with gain and 85.1% with no change. As expected, the threshold value of > 2mm resulted in fewer sites with attachment level change (Table 3): these values were 6.1% loss, 0.4% gain, and 93.5% with no change. For both threshold values, the percentage of sites with attachment loss increased as the study progressed. Since this study was an examination of disease progression, attention was mainly focused on those sites with attachment loss.

Distinct differences were noted in the distribution of attachment loss within the three subject groups. By nine months (Figure 2) it was evident that both the untreated and the treated/no maintenance groups exhibited similar levels of attachment loss, while the treated/maintenance group exhibited proportionately less. Using >1 mm as the threshold for change, 13.0% of the untreated sites, 13.2% of the treated/no maintenance sites, and 9.9% of the treated/maintenance sites lost attachment, while the values using a threshold of > 2mm were 6.0%, 6.2%, and 3.6% respectively.

When attachment loss was examined as a function of the baseline probing depth, two different patterns were again noted. In both the untreated and treated/no maintenance groups (Tables 4 and 5), sites with probing depths

Table 2: Overall Attachment Level Change > 1 mm. For each time interval, the percentage of sites with attachment loss, attachment gain, and no change are listed.

INTERVAL	LOSS	NO CHANGE	GAIN
3 MONTH	6.7%	91.2%	2.1%
6 MONTH	9.6%	88.7%	1.4%
9 MONTH	13.5%	85.1%	1.4%

Table 3: Overall Attachment Level Change $\geq$ 2 mm. For each time interval, the percentage of sites with attachment loss, attachment gain, and no change are listed.

INTERVAL	LOSS	NO CHANGE	GAIN
3 MONTH	2.5%	97.1%	0.4%
6 MONTH	3.8%	94.8%	0.4%
9 MONTH	6.1%	93.5%	0.4%

Figure 2.

Group comparison of Attachment Loss at 9 months. For the Untreated group, 13.0% of all sites lost > 1mm of attachment and 6.0% lost ≥ 2mm. These values for the Treated/No Maintenance group were 13.2% > 1mm and 6.2% ≥ 2mm. In the Treated/Maintenance group, 9.9% of all sites had attachment loss > 1mm and 3.6% ≥ 2mm.

ATTACHMENT LOSS AT 9 MONTHS

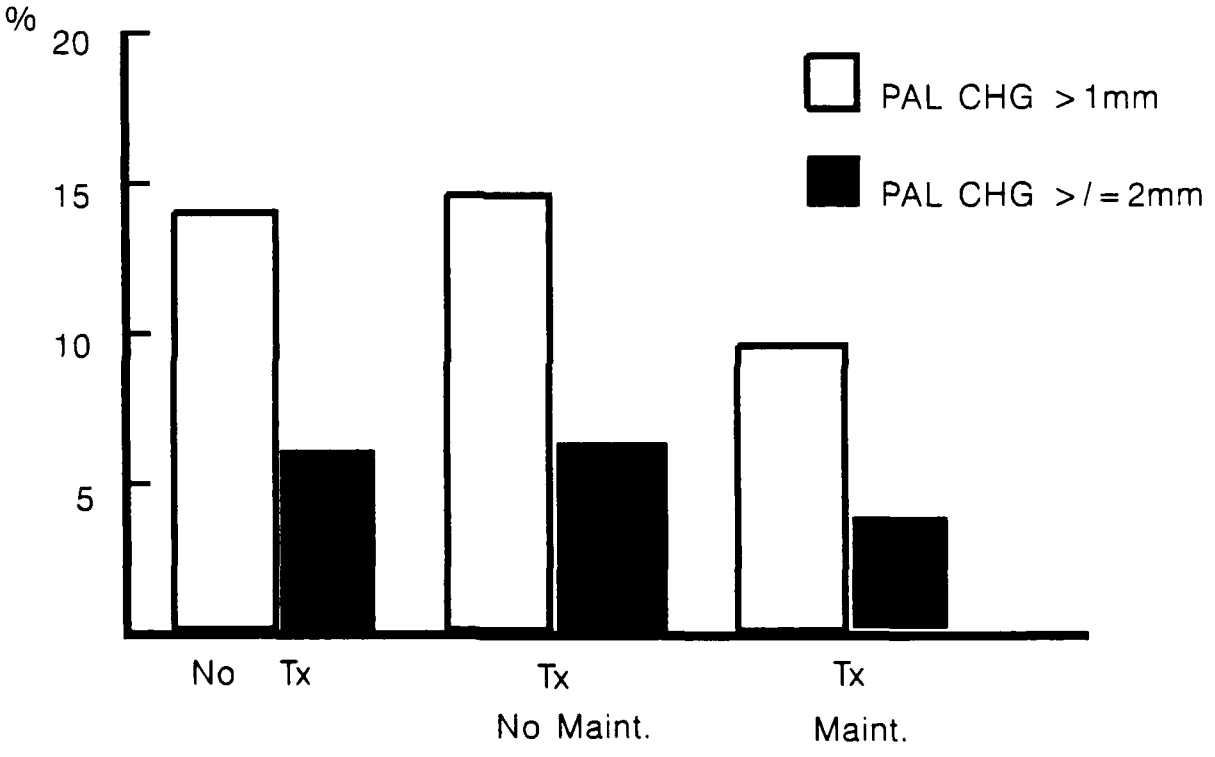


Table 4: Attachment Loss vs. Baseline Pocket Depth (Untreated Subjects). The percentage of sites with initially deep and shallow probing depths (PD) that experienced subsequent attachment loss is listed.

BASELINE PD	ATTACHMENT LOSS > 1 mm	ATTACHMENT LOSS ≥ 2 mm
≤ 3 mm	6.3%	2.6%
> 3 mm	14.6%	7.0%

Table 5: Attachment Loss vs. Baseline Pocket Depth (Treated/ No Maintenance Subjects). The percentage of sites with initially deep and shallow probing depths (PD) that experienced subsequent attachment loss is listed.

BASELINE PD	ATTACHMENT LOSS > 1 mm	ATTACHMENT LOSS ≥ 2 mm
≤ 3 mm	10.7%	4.5%
> 3 mm	35.6%	20.7%

Table 6: Attachment Loss vs. Baseline Pocket Depth (Treated/ Maintenance Subjects). The percentage of sites with initially deep and shallow probing depths (PD) that experienced subsequent attachment loss is listed.
*

BASELINE PD	ATTACHMENT LOSS > 1 mm	ATTACHMENT LOSS ≥ 2 mm
≤ 3 mm	10.2%	4.1%
> 3 mm	8.8%	0.0%

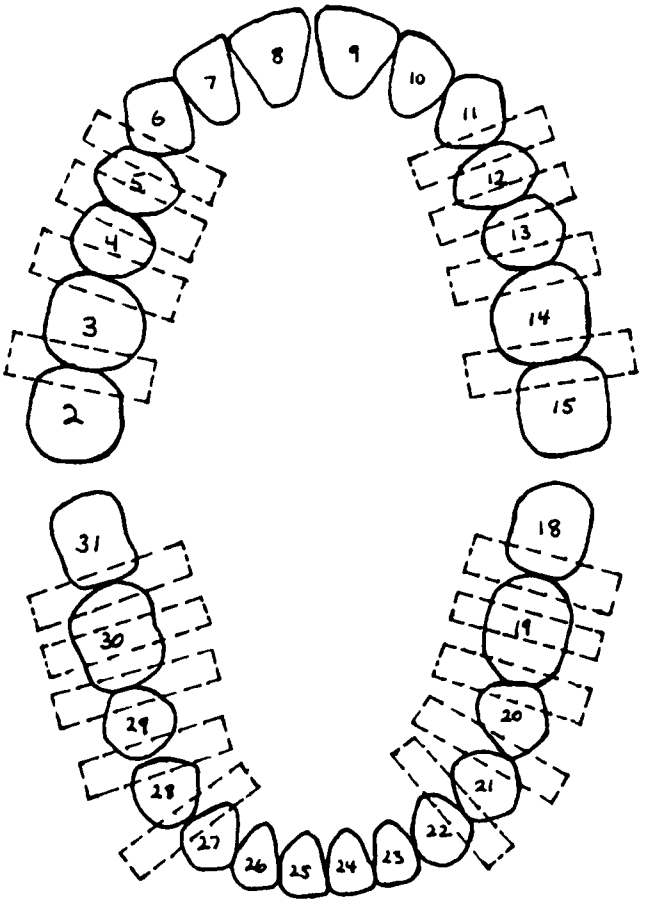
initially greater than 3 mm were seen to undergo further loss of attachment at a higher rate than were sites initially 3mm or less. Using >1mm as the threshold for significant change, 6.3% of shallow sites and 14.6% of deep sites in the untreated group experienced additional attachment loss, while these values for the treated/no maintenance group were 10.7% and 35.6%. Using > 2mm as the threshold, 2.6% of shallow sites and 7.0% of deep sites lost attachment in the untreated group, while 4.5% of shallow sites and 20.7% of deep sites lost attachment in the treated/no maintenance group. In contrast, shallow sites in the treated/maintenance group appeared more prone to attachment loss (Table 6). In that group, 10.8% of shallow sites and 8.8% of deep sites had additional attachment loss of > 1mm, while 4.1% of shallow sites and 0.0% of deep sites had attachment loss $\geq$ 2mm.

C. CADIA Analysis of Dental Radiographs.

As opposed to the attachment level measurements, radiographic analysis of density change was not recorded at individual sites. Due to the two dimensional nature of radiographs and the fact that mesial and distal probing measurements were recorded on both facial and lingual surfaces, one area of alveolar bone available for density analysis could correspond to as many as four probing sites (Figure 3). For this reason, it was necessary to report radiographic change in terms of these 'radiographic complexes' rather than by sites. From the radiographs of all subjects, 281 of these complexes were identified. Fifty-nine of the 281 were associated with attachment loss $\geq$ 2mm at nine months, while the other 222 were not. Bone density changes were recorded in computer assisted densitometric image analysis (CADIA) units,

Figure 3.

Radiographic Complexes. Due to the 2 dimensional nature of radiographs and the fact that probing measurements were recorded on both facial and lingual surfaces, one area of alveolar bone available for density analysis could represent as many as four probing sites. Anterior sites were not available for density analysis due to the method of standardizing radiographs with a cephalostat.



RADIOGRAPHIC COMPLEXES

Table 7: Density change at alveolar bone sites with attachment loss $\geq$ 2mm. Fifty-nine of the 281 total radiographic complexes were associated with at least one probing site that experienced $\geq$ 2mm attachment loss during the 9 month study period. Alveolar bone density change is listed as density loss (degeneration), density gain (regeneration), or no change. For bone sites with density loss, a breakdown of the magnitude of degeneration is listed.

NET DENSITY CHANGE*	NUMBER OF SITES	PERCENTAGE
Regeneration or No change: (0 or +)	27	45.8%
Degeneration: (< 0)	32	54.2%
($\leq$ -3)	19	32.2%
($\leq$ -6)	12	20.3%

* Density Change recorded in CADIA units

Table 8: Density change at alveolar bone sites with no significant attachment loss. 222 of the 281 total radiographic complexes were associated with no attachment loss $\geq$ 2mm during the 9 month study period. Alveolar bone density change is listed as density loss (degeneration), density gain (regeneration), or no change. For bone sites with density loss, a breakdown of the magnitude of degeneration is listed.

NET DENSITY CHANGE*		NUMBER OF SITES	PERCENTAGE
Regeneration or No change:	(0 or +)	127	57.2%
Degeneration:	(< 0)	95	42.8%
	($\leq$ -3)	43	19.4%
	($\leq$ -6)	28	12.6%

* Density Change recorded in CADIA units

which as mentioned previously were derived from both the area and magnitude of density change within each observed area of alveolar bone.

Density change at the 281 radiographic complexes was identified as either degeneration, regeneration, or no change. Of the 59 radiographic complexes associated with attachment loss $\geq$ 2mm (Table 7), 54 2% were found to have degeneration at the alveolar crest, while 45.8% were found to have either regeneration or no change. Of the 222 complexes without significant attachment loss (Table 8), 42.8% were found to have degeneration compared to 57.2% with regeneration or no change. Radiographic complexes associated with attachment loss also had a proportionately higher percentage of sites with increasing magnitudes of degeneration.

D. Correlation between Clinical and Radiographic Data.

The next aspect of the data analysis was to determine whether or not a correlation existed between density change and attachment level change (Table 9). The 59 radiographic complexes with $\geq$ 2mm of attachment loss experienced a mean degeneration in CADIA units of -6.12, as compared to a mean CADIA value of -2.36 for those complexes without significant attachment loss. When analyzed by a T test, these 2 groups were found to be significantly different at $P < .05$. When 9 month attachment level change was plotted against bone degeneration at 3 and 6 months, there was no difference between sites with and without attachment loss.

The final correlation between density change and attachment level change

was determined by the number of sites with attachment loss within each radiographic complex (Table 10). Since each complex could include up to 4 probing sites, there were from 0 to 4 sites with attachment loss > 2 mm. The mean degeneration at the alveolar crest increased from -2.36 CADIA units at radiographic complexes with no attachment loss to -14.90 at the one complex with attachment loss at three sites.

Table 9: Correlation between Density Loss (Degeneration) and Probing Attachment Loss. Of the 281 radiographic complexes observed in all subjects, 59 were associated with $\geq$ 2mm attachment loss and 222 were not. Listed below is the mean degeneration observed in both groups.

	NUMBER OF RADIOGRAPHIC COMPLEXES	MEAN DEGENERATION	RANGE
ATTACHMENT LOSS $\geq$ 2mm	59	-6.12*	(-57.7 - 0)
NO SIGNIFICANT ATTACHMENT LOSS	222	-2.36*	(-45.3 - 0)

* - Degeneration recorded in CADIA units.

* - When the above means were subjected to a T- test, it was found that there was a significant difference at $p < .05$.

Table 10: Degeneration vs. the number of sites with attachment loss in each radiographic complex. Each radiographic complex could include up to four probing sites.

NUMBER OF SITES WITH ATTACHMENT LOSS	NUMBER OF RADIOGRAPHIC COMPLEXES	MEAN DEGENERATION*
0	222	-2.36
1	45	-4.40
2	13	-11.27
3	1	-14.90
4	0	-

* - Degeneration recorded in CADIA units

IV. Discussion and Summary

The ability to accurately monitor disease progression remains one of the primary goals of periodontal research. Both clinical experience and a large number of published reports have established the longitudinal assessment of attachment level change as the 'gold standard' for disease progression. While radiographic changes most certainly occur with the progression of periodontitis, the appearance of bone tissue in radiographs has never been found to strongly correlate with the clinical parameters of disease. In recent years, new advances in computer technology have allowed more accurate detection of slight changes in alveolar bone. In this study, density changes at the alveolar crest in subjects with a history of periodontitis were compared to attachment level changes at the same sites. In this manner, the relative frequency of disease progression was examined using both new and conventional techniques.

In an effort to increase the accuracy of the attachment level data, duplicate probing measurements were made at three month intervals using stents as fixed reference points. The stent design used in this study was a variation of the conventional vacuform matrix used to make temporary crowns. In addition to vacuum adaptation to the study cast, the matrix material was pressure molded to the cast at the same time. The end result was a very thin, well adapted stent that retained its shape and did not have to be held in place by finger pressure. This stent was thin enough to allow the probing measurement to be made at a near vertical angulation into the interdental space, while thicker stents tend to increase the horizontal angulation and

may therefore increase probing error.

Using this methodology to obtain clinical measurements, attachment loss $\geq$ 2mm was noted at 6.0% of untreated sites, 6.2% of treated/no maintenance sites, and 3.6% of treated/maintenance sites over the nine month period. These values are somewhat higher than those of some previous studies. Haffajee et al (1983) noted significant attachment loss at 2.8% of sites in a group of 22 untreated patients monitored over one year. Harley et al (1987) observed that 3.7% of sites in a group of 10 untreated patients lost $\geq$ 2mm of attachment over 12 weeks. Lindhe et al (1983) reported that 3.2% of 3210 sites in 36 untreated subjects had > 2mm additional attachment loss over one year. The findings of the present investigation are in line with those of Goodson (1982), who noted a significant increase in attachment level measurements in 5.7% of sites in 22 subjects with untreated disease. In contrast, a higher level of disease activity was noted by Jenkins et al (1988), who found that 9.8% of all sites in a group of advanced periodontitis patients lost at least 2 mm attachment over a one year period. The differences noted between various studies may be attributed to differences in the significance level for change, the time interval over which measurements were made, individual subject variation, differences in probing technique, and perhaps differences in patient populations. The lower percentage of sites with attachment loss in the treated/maintenance group is consistent with previous reports of the importance of maintenance therapy in the long term management of periodontitis (Becker et al 1984, Lindhe and Nyman 1984).

One interesting finding in the attachment level data dealt with

attachment level change as function of baseline pocket depth. While both clinical impressions and published reports are somewhat mixed on this subject, perhaps the most frequently cited report is that by Lindhe et al (1983), which stated that deeper sites appear no more prone to additional attachment loss than are shallow sites. In the present study, the data for 2 of the 3 subject groups tended not to support this finding. For example, in the 10 untreated subjects, 7.0% of all sites initially deeper than 3 mm lost at least 2 mm of attachment, compared to 2.6% of sites initially 3mm or less. This same trend was observed in the treated/no maintenance group, but not in those patients undergoing active maintenance care. In that group, the shallower sites lost more attachment, a finding which may be expected if periodic instrumentation was performed at shallow sites as part of maintenance therapy. The high percentage of sites with further attachment loss in the treated/no maintenance group confirms previous reports that periodontal therapy with no follow-up care is of little long term benefit (Nyman et al 1977).

Radiographic density change noted within the previously described radiographic complexes demonstrated a large number of sites with density loss (degeneration) over the nine month period regardless of the degree of attachment loss. There was a slightly higher percentage of sites with degeneration in the 59 complexes with attachment loss, but perhaps more significant was the higher percentage of sites with an increased magnitude of degeneration as recorded in CADIA units. These results differ with those of Hausmann et al (1986), who in a six month study of untreated patients found density loss at 9% and density gain at 4% of interdental sites. The authors

of this study used their data to support the site specific, episodic progression theory of periodontal disease.

There are several possible explanations for the high percentage of sites with density loss in this study. First, since the image analysis system was programmed with high sensitivity to density change, it is possible that small variations caused by background noise within the CADIA system were recorded as true density change. This possibility may be unlikely since changes were consistently recorded as degeneration rather than random gains and losses of bone density. Second, it must be remembered that each of these subjects had a documented susceptibility to periodontitis and only three received maintenance therapy during the study period. It is possible that this degeneration is indicative of small increments of continuing disease activity that are not reflected in attachment level measurements. Third, it is possible that these density measurements have simply recorded normal patterns of density change within alveolar bone that are totally unrelated to disease progression.

The last aspect of the data analysis concerned the correlation between density change and attachment level change during the study period. The most popular theory concerning this relationship was proposed by Goodson (1984) in a study comparing attachment loss to changes in bone height. He determined that attachment loss was followed in several months by bone loss at the alveolar crest. Using the parameter of density change rather than bone height, the data from the present study suggests that differences exist between sites with and without attachment loss at the same nine month

interval. Of the 281 sites available for bone density analysis, sites both with and without significant attachment loss exhibited a mean degeneration of alveolar bone at nine months; however, sites with attachment loss experienced significantly greater degeneration. It should be noted, however, that the CADIA values within both groups varied widely, raising questions about the site specific relevance of this density change.

Reports by Jeffcoat et al (1980, 1982, 1986) have suggested that there may be a predictive value to bone changes at the alveolar crest. Based on their studies, high uptake of the bone seeking radiopharmaceutical 99m-Tc-MDP at the alveolar crest was an accurate detector of disease activity 6 months later. Techniques such as 99m-Tc-MDP measure alterations in bone metabolism rather than density, and may therefore yield results that are quite different from radiographic assessment. In order to test the predictive ability of alveolar bone changes in the present investigation, 9 month attachment level data was plotted against alveolar bone degeneration at 3 and 6 months. Examination of this data revealed no differences between complexes with and without 2mm of attachment loss. This casts some doubt on the ability of bone density change, as assessed in this study, to predict future episodes of attachment loss.

The final correlation between attachment loss and density change was the relationship between degeneration and the number of sites with attachment loss within each radiographic complex. As noted in table 10, density loss increased as more sites were affected. Statistically, there were significant differences between complexes with 0 or 1 attachment loss site and those with

loss at 2 or 3 sites. Since there were fewer complexes with multiple attachment loss sites, this data must be interpreted with some caution.

In conclusion, this study has longitudinally evaluated both clinical and radiographic data from a group of patients with a history of periodontitis, with special emphasis on the investigation of a correlation between these two parameters. While this study has demonstrated a correlation between attachment loss and density loss at the alveolar crest, there appears to be a wide range of variability at individual sites. For this reason, even though densitometric analysis of radiographs has proven useful in quantitating site specific changes in alveolar bone, at this time it appears to have limited usefulness in either identifying or predicting sites with attachment loss.

This study also underlines the major problem that continually hampers longitudinal studies of periodontitis; that is, investigations of new techniques to identify disease progression are made difficult by comparison to the inadequate 'gold standard' of probing attachment level change. Until this problem is resolved, studies such as this one will continue to yield information whose potential importance is diluted by comparison to inadequate standards.

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Vita

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